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Revealing of Hormonal Characteristics of Phenotype A Polycystic Ovarian Syndrome

Zainab G. Falh[1], Basil O. Saleh[1], Afraa M. AL- Naddawi[2]

[1] Department of Clinical Biochemistry, College of Medicine, University of Baghdad, Baghdad, Iraq.

[2] Department of Obstetrics & Gynecology, College of Medicine, University of Baghdad, Baghdad, Iraq.

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Correspondence: Zainab G. Falh
Email: Zz7578942@gmail.com

Website: https://djm.uodiyala.edu.iq/index.php/djm

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Abstract

Background: Phenotype A also referred the classic PCOS phenotype, represents the most severe clinical form as it encompasses all three diagnostic features clinical and/or biochemical hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM). it is more commonly found in subjects identified in clinical populations.

Objectives: To investigate the hormonal changes in phenotype (A) of polycystic ovary syndrome (PCOS).

Patients and Methods: This study was carried out at Department of Biochemistry, College of Medicine, University of Baghdad. Investigations included serum measurements of anti-müllerian hormone (AMH), free testosterone, inhibin B, by using enzyme linked immunosorbent assay (ELISA) technique, prolactin, luteinizing hormone (LH), follicle stimulating hormone (FSH) were measured by TOSOH technique. The ratio of LH/FSH was calculated.

Results: The mean (±SEM) value of serum free testosterone levels of phenotype (A) PCOS was significantly higher than that of controls (p=0.001). However, the mean (±SEM) value of AMH levels of phenotype (A) PCOS was higher than that of control women, but did not reach the significant level (p=0.06). The mean (±SEM) values of LH levels and LH/FSH ratio, of phenotype (A) PCOS were significantly higher than those of control women (p=0.03, p=0.001, respectively). In addition, significant correlations were observed among the studied hormones in phenotype (A) PCOS.

Conclusion: Phenotype A is the predominant one of PCOS phenotypes and is associated with highest serum AMH, free testosterone and obesity. The pattern of hormonal changes and correlations among them may shed light on the new pathophysiology of the phenotype A PCOS and may aid in treatment strategy.

Keywords: Anti-müllerian hormone, Free testosterone, Inhibin B, LH/FSH ratio, Polycystic ovary syndrome.

Introduction

Polycystic ovarian syndrome (PCOS) is one of the rapidly emerging endocrine health issues affecting women of reproductive age worldwide (1,2). PCOS can be identified by three main symptoms: irregular menstrual cycles, hirsutism (HA), and the presence of polycystic ovarian morphology (PCOM) thus named in the USA (3). In 1935 Stein and Leventhal described this illness in their publication entitled “Amenorrhea Associated with Bilateral Polycystic Ovaries” (4). They discovered that PCOM has correlations with a number of other significant and subtle symptoms. Their investigation

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identifies many signs including amenorrhea, infertility, masculinizing and oligomenorrhea alterations such as HA, goiter, and obesity (4). The precise role of biological pathways in these anomalies remains intricate and uncertain (5). Identifying probable molecular pathways and interacting processes that contribute to the development of PCOS might give valuable information for the development of diagnostic and therapeutic targets to regulate metabolic abnormalities in people with PCOS (6). The Rotterdam criteria delineate four primary phenotypes which are determined by the clinical manifestations and symptomatic findings of PCOS (7). In order to get more favorable results, it is crucial to identify the phenotypes in patients with PCOS using the right methods (8). Phenotype A was characterized by the presence of oligomenorrhea- anovulation, (HA), and polycystic ovaries (PCOM) on ultrasound. Phenotype B was identified by the presence of oligomenorrhea- anovulation and HA. Phenotype C was described as having HA and polycystic ovaries on ultrasound. Phenotype D was diagnosed as having oligomenorrhea-anovulation and polycystic ovaries on ultrasound (9). When comparing these four phenotypes, it is seen that phenotypes A and B exhibit symptoms with greater severity (3, 10). Phenotype D is recognized as the least severe variant, whereas phenotype C is intermediate in severity, falling between phenotypes A-B and D (3, 10). In order to be diagnosed with PCOS, a patient must exhibit at least two out of the three primary symptoms, as outlined by the Rotterdam criteria (3). Hyperandrogenism may be classified into two primary categories: hyperandrogenism and a greater, biochemical and clinical (10). hyperandrogenism and a greater prevalence of PCOM (11). Biochemical hyperandrogenism is characterized by slightly to moderately increased amounts of androgenic precursors, including dehydroepiandrosterone sulfate (DHEAS), androstenedione, and biologically active androgens such as testosterone (12). HA is the primary clinical expression of hyperandrogenism, whereas acne and alopecia are considered secondary clinical signs (10, 12). Exposure to androgens intensifies the symptoms of PCOS. Exposure to elevated amounts of prenatal androgens is another factor that might contribute to a higher incidence of developing PCOS (13). Hyperandrogenism may also be caused by aromatase insufficiency or abnormalities (14). The absence of functional aromatase enzyme results in a decrease in the conversion of testosterone to estradiol, causing an accumulation of androgens (14). Anti-Müllerian hormone (AMH) serves as a diagnostic marker for PCOS. AMH is accountable for the process of follicle maturation and development. Excessive production of AMH may lead to dysfunction of the ovaries (15). AMH and its receptor variants with reduced functionality account for 6.7% of PCOS cases (16). A beta-B subunit and an alpha subunit are joined to form the glycoprotein known as inhibitor B. It is categorized as belonging to the superfamily of transforming growth factor-B. It is well known that the non-steroidal hormone, which is generated by granulosa cells in developing follicles, may suppress the follicle-stimulating hormone (FSH) (17). Previous studies suggest that inhibin B may be useful in evaluating ovarian aging, detecting premature ovarian failure (POF) or premature ovarian insufficiency, measuring ovarian function in cancer survivors, and predicting results of assisted reproductive technology (17). The raised LH to FSH ratio is significant due to an augmented occurrence of gonadotropin hormone-releasing this hormone pulses, indicating the presence of the disease (18, 19). Scenario mostly arises from an overabundance of LH and ovarian androgens, or the excessive stimulation of LH (11, 20-22). The aim of the study is to evaluate the role of serum levels of anti- Mullerain hormone (AMH),

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inhibin B and free testosterone in differentiation of different phenotypes (A) of polycystic ovary syndrome (PCOS).

Patients and Methods

Study design: The case-control study was performed at the Department of Biochemistry, College of Medicine, University of Baghdad and at Baghdad Teaching Hospital, Medical City, Ministry of Health, Baghdad, Iraq, during the period from November 2023 to March 2024. The study comprised 62 women, aged 18-40 years, 42 of whom were diagnosed to have had phenotype (A) PCOS by a consultant Gynecologist and 20 apparently healthy women as controls. This study gynecologist was approved to be carried out by the scientific and ethical committees of the Department of Biochemistry, College of Medicine, University of Baghdad. Ethical approval was also obtained from Baghdad Teaching Hospital, Medical City, Ministry of Health. Vocal consent was obtained from each of the included women in order to participate in this study (23).

Inclusion criteria: for women with phenotype (A) PCOS involve having the three criteria of the 2003 Rotterdam Consensus including: (1) oligomenorrhea- anovulation, (2) hyperandrogenism (and/or biochemical results), and (3) polycystic ovaries (identified by ultrasound of the women being 18-40 years.

Exclusion criteria: included those, women who were taking oral contraceptives during blood draws and other diagnoses mimicking PCOS (i.e. hyperprolactinemia, premature ovarian failure, congenital adrenal hyperplasia), thyroid gland dysfunctions, Cushing disease, liver disease, kidney disease and cancers. Five millimeters (ml) from the peripheral vein were aspirated from each PCOS and control woman, left to clot for 15 minutes, and then centrifuged for 10 minutes at 2500 rpm. The separated serum was till the day of measurements. Serum investigation stored at -45°C for measurements of AMH, free testosterone, and inhibin B.

Free testosterone, anti-müllerian hormone (AMH) and inhibin B: Semiautomatic Enzyme Linked Immunosorbent Assay (ELISA) Reader Huma, Reader by Human Diagnostics German Company, Washer (COMBIWASH) by HUMAN Germany Company. The principle of ELISA technique is based on sandwich, technique using biotin double antibody sandwich technology to assay the free testosterone, anti-müllerian hormone (AMH) and Inhibin B. Add hormones to the wells, which are pre-coated with its monoclonal antibody and then incubate. After that, add anti hormones antibodies labeled with biotin to unite with streptavidin-HRP, which forms immune complex. Remove unbound enzymes after incubation and washing. Add substrate A and B. Then the solution will turn blue and change into yellow with the effect of acid. The shades of solution and concentration of Human Free testosterone or AMH are positively correlated.

Serum LH, FSH, and prolactin measurements: Assay by Tosoh AIA-2000 Automated Immunoassay, Japanese principle was used. The ST AIA-PACK test is a two-site immunoenzymometric assay which is performed entirely in the ST AIA-PACK test cups serum present in the test sample is bound with monoclonal antibody immobilized on a magnetic solid phase and enzyme-labeled monoclonal antibody in the test cups. The magnetic beads are washed to remove unbound enzyme-labeled monoclonal antibody and they are then incubated with a fluorogenic substrate 4-methylumbelliferyl phosphate (4MUP). The amount of enzyme-labeled monoclonal antibody that binds to the beads is directly proportional to the serum concentration in the test sample. A standard curve is constructed and unknown sample concentration are calculated using this curve. The LH/FSH ratio was computed. Body mass index (BMI) was calculated using the international equation:

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BMI (kg/m2) = weight (kg) / height (m2).

Statistical Analysis

SPSS 25 software was used. The mean and standard error of the mean (SEM) were used to interpret the statistical results. A t-test was used to evaluate the difference in the mean level of numeric data between two variables. Area under the curve (AUC) and Receiver Operating Characteristic (ROC) curves were analyzed, and cutoff values, sensitivities, and specificities were derived to differentiate among the women studied. Pearson correlation regression (r) was used to evaluate the relationship between numerical data. The significance level was chosen at $p < 0.05$.

Results

Demographic characteristics: Table 1 presents the mean (±SEM) values for age and BMI across the study groups. The mean age of phenotypes (A) was significantly lower than that of controls (25.76±0.65; p=0.01). The mean BMI for phenotype (A) PCOS was significantly higher than that of the control (31.15±1.04, p=0.001).

Table 1. Mean (±SEM) values of age and body mass index of phenotype (A) polycystic ovarian syndrome women and controls.

Parameter	Phenotype A PCOS (n=42)	controls (n=20)
Age (year)	25.76±0.65 •	29.95± 1.42
BMI (Kg/m^2)	31.15±1.04 •	25.05± 0.64
ANOVA and t-test reveals: ●significant decrease in mean values of age of phenotypes A PCOS (p=0.01) and significant increase in BMI in phenotypes A PCOS (p=0.001) than in controls.		

Hormonal result: Table 2 shows the mean (±SEM) values of serum AMH, free testosterone (FT), and inhibin B concentrations of phenotype (A) PCOS and control women. The mean value of serum AMH levels of phenotype (A) was on the border of a significant level in comparison with control women (5.52±0.73, p=0.06). The mean value of FT levels of phenotypes (A) PCOS was significantly higher than that of control women (0.79±0.10, p=0.001). The mean value of inhibin B levels of phenotype (A) was lower than that of control women (50.46± 7.12), but did not reach a significant level.

Table 2. Mean (±SEM) values of Anti-müllerian hormone, Free testosterone, and inhibin B of phenotype (A) polycystic ovarian syndrome women and controls.

Parameter	Phenotype A PCOS (n=42)	Controls (n=20)
AMH (ng/ml)	5.52±0.73•	3.46±0.22
Free Testosterone (nmol/l)	0.79±0.10••	0.37±0.02
Inhibin B (pg/ml)^{NS}	50.46± 7.12	57.68± 2.07
ANOVA and t-test revealed: ● borderline significant increase in AMH in phenotype A PCOS than in controls (p=0.06), ●● significant increase in free testosterone in phenotype A than in controls (p=0.001). NS: non-significant difference in inhibin B.		

Table 3 shows the mean (±SEM) values of serum LH, FSH, prolactin and the LH/FSH ratio of phenotype A PCOS and control women. The mean value of LH levels of phenotype A PCOS was significantly higher than that of control women (7.12±0.76, p=0.03). The mean value of LH/FSH ratio were significantly elevated in phenotypes A PCOS when compared to controls (1.06± 0.11, p=0.001). However, the mean (±SEM) values of FSH and prolactin did not differ significantly between phenotype A PCOS and control women.

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Table 3. Mean (±SEM) values of luteinizing hormone (LH), follicle stimulating hormone (FSH), prolactin, and LH/FSH ratio of phenotype a polycystic ovarian

Parameter	Phenotype A PCOS (n=42)	controls (n=20)
LH (µIU/ml)	7.12±0.76♠	4.59±0.38
FSH (µIU/ml)^{NS}	7.71± 0.65	8.44± 0.49
Prolactin (ng/ml)^{NS}	13.30± 1.29	9.90± 0.69
LH/FSH ratio	1.06± 0.11♠♠	0.58± 0.05
ANOVA and t-test reveals: ♠significant increase in LH in phenotypes A PCOS than in controls (p=0.03). ♠♠ Significant increase in LH/FSH ratio in phenotypes A PCOS than in control women (p=0.001). NS: non-significant differences in FSH and prolactin.		

Correlations: In phenotype A PCOS group, there were significant positive correlations between inhibin B of AMH (r=0.84, p=<0.001) as shown in Figure 1.

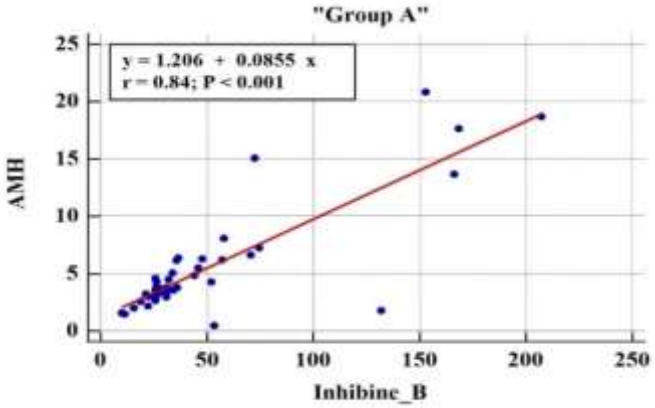

Figure 1. Correlation between AMH (ng/ml) and inhibin B (pg/ml) of Phenotype A.

In phenotype A PCOS group, there were significant positive correlations between inhibin B and free testosterone levels (r=0.53, p=<0.001, Figure 2).

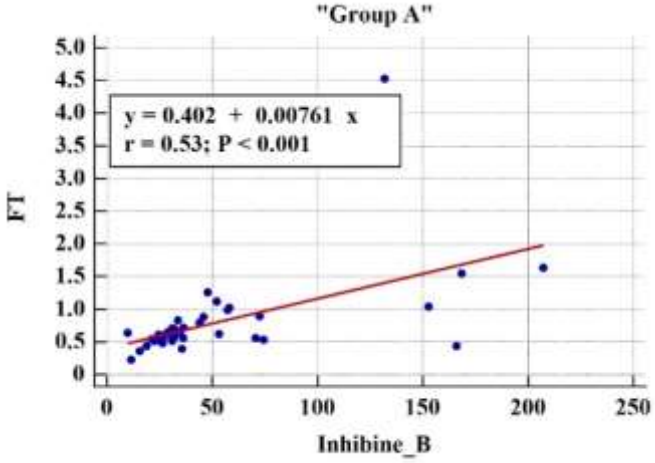

Figure 2. Correlation between free testosterone (nmol/l) and inhibin B (pg/ml) of Phenotype A.

There was also significant positive correlation between LH and LH/FSH ratio (r=0.80, p=<0.001, Figure 3).

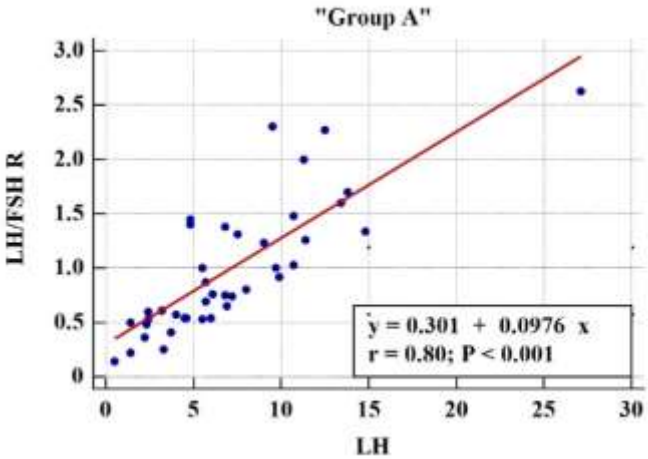

Figure 3. Correlation between LH (µIU/ml) and LH/FSH ratio of Phenotype A.

Significant positive correlation between FSH with age of PCOS women (r=0.33, p=0.03, Figure 4).

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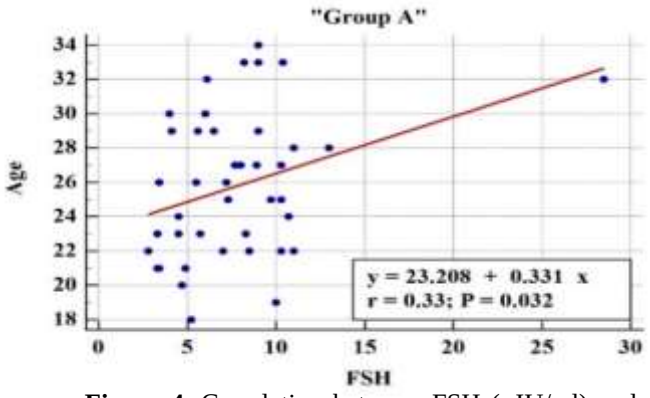

Figure 4. Correlation between FSH (µIU/ml) and age (year) of Phenotype A.

Receiver operating characteristic (ROC) and the area under curve: Study revealed that serum free testosterone at (cutoff = 0.47 ng/ml) was the best test in differentiation of phenotype A. PCOS from control women with AUC value 0.939 (sensitivity= 88.1 % and specificity= 100 %, figure5). Inhibin B has AUC=0.774 at cutoff value (36.468 pg/ml) with sensitivity=64.29 % and specificity =100 % in such differentiation.

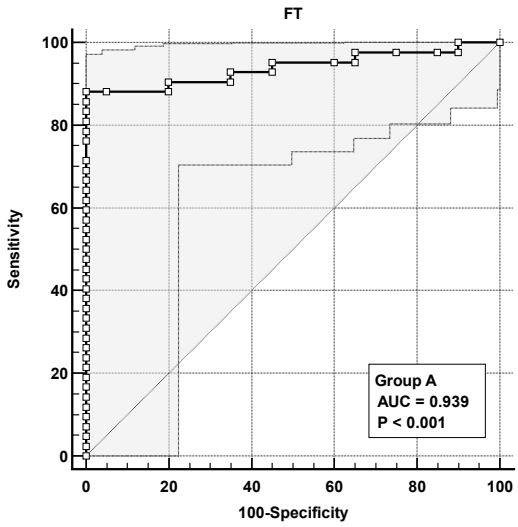

Figure 5. Receiver operator characteristic (ROC) and area under curve (AUC) study of phenotype A.

Discussion

The present study is concerned with phenotype A PCOS, because it represents the majority of one of the phenotypes PCOS (24-26). Phenotype A PCOS women have the main three criteria and hence its highest prevalence in lots of studies. The fact that it represents the bases to diagnose PCO. The mean age value of the entire group of PCOS women in the present study was found to be 25.76 years and that of BMI 31.15 Kg/m2 (Table 1). Mehra et al. found the mean age of their PCOS women was 24.0 years (27), and Carmina and Lobo found the mean age of their PCOS women was 24.2 year (28). However, Wiweko et al. observed that the mean age of their PCOS patients was 29.6 years their BMI was 25.7 Kg/m2 (29), and Malhotra et al. found that their PCOS patients were 28.2 years old and their BMI was 26.33 Kg/m2(30). These differences may be due to the population race, lifestyle, and their type of diet. The present study found a significant increase in the mean value of free testosterone level in phenotype A PCOS (Table 2), in consistency with that reported by Mehra et al. who found the mean values of free and total testosterone levels were significantly higher in phenotype A (27). These authors reported that increased serum testosterone levels in PCOS women are associated with excess visceral fat. Gürsu et al. observed that obesity and hyperandrogenism were more common in phenotype A PCOS, suggesting a higher risk of adverse metabolic effect outcomes in this phenotypic group (22). The current study also found that the mean value of serum AMH levels was higher in phenotype A than in control (Table 3). This finding is in harmony with that reported by Amini et al. who found the mean value of AMH levels was highest in phenotype A PCOS (31). Similarly, Wiweko et al. and Jamil et al. also demonstrated that the mean value of AMH was higher in phenotype A (29, 32). Wiweko et al. also reported that the level of AMH has been shown to have a correlation with oligo-anovulation (29), and those PCOS women who were anovulatory had a 12-fold greater amount of AMH than the ovulatory women (33). Önal and Öztürk found that AMH levels were greatest in phenotypic A and lowest in the control group (34). The current research found the mean value

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of serum Inhibin B of phenotype A lower than controls (Table 3), which is in agreement with that found by Hussein et al. and Obaid et al, who found serum Inhibin B levels were significantly lower in PCOS women than in controls (35, 36) However, Fazil et al. did not find any significant variation in serum Inhibin B between their PCOS women and controls (37). By contrast, Farman et al. found that serum inhibin B levels of PCOS women were significantly greater than the controls (38). An essential function of inhibin B is to regulate ovarian function (39). Wen et al. found that Inhibin B was positively correlated with AMH and testosterone (17). The current study also found that serum LH level and LH/FSH ratio were highest in phenotype A (Table 3) which are in agreement with those observed by Gürsu et al. (22). Sharmin et al. indicated that the most common phenotypic and severe form of PCOS was phenotype A. These authors concluded that phenotype A PCOS had significant biochemical hyperandrogenism, abnormal LH levels, and a changed LH/FSH ratio (40). Jamil et al. found that genotype A PCOS had significantly higher levels of total testosterone and the LH/FSH ratio (32). Gürsu et al. and Önal and Öztürk did not find any significant differences in serum prolactin among and between PCOS phenotypes and controls (22, 34).

Conclusion

The pattern of hormonal changes and correlations among them may shed light on the on the new pathophysiology of the phenotype A PCOS and may aid in treatment strategy. Therefore, it was recommended that it is important to study the prognostic role of AMH, Inhibin B and free testosterone in phenotype A of PCOS before and after their treatment.

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Ethical clearance: The entire work was permitted by ethical clearance Committees of local authorities (code no. 191). All participants provided informed consent, and the research was conducted in accordance with the ethical principles set out in the 1975 Declaration of Helsinki. The authors declare no potential conflicts of interest related to the present research.

Conflict of interest: None.

Use of Artificial Intelligence (AI): The authors state they did not use any generative AI tools for creating or editing the manuscript's language.

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الملف الهرموني للنمط الظاهري (أ) متلازمة المبيض المتعدد الكيسات

[١] زينب جهاد فالح ،[١] باسل عويد محمد صالح ،[٢] عفراء محجوب النداوي،

الملخص

الخلفية: يُعرف النمط الظاهري (أ) أيضًا بالنمط الظاهري التقليدي لمتلازمة تكيس المبايض، وهو الشكل السريري الأكثر شدة، إذ يشمل جميع السمات التشخيصية الثلاث: فرط الأندروجين السريري و/أو الكيميائي الحيوي (**HA**)، وخلل التبويض (**OD**)، ومورفولوجيا المبيض المتعدد التكيسات (**PCOM**). وهو أكثر شيوعًا لدى الحالات التي تم تشخيصها ضمن الفئات السريرية.

الأهداف: دراسة التغيرات الهرمونية في النمط الظاهري (أ) لمتلازمة تكيس المبايض (**PCOS**).

المرضى والطرق: أُجريت الدراسة في قسم الكيمياء الحيوية، كلية الطب، جامعة بغداد. شملت الفحوصات قياسات مصل هرمون مضاد مولر (**AMH**)، والتستوستيرون الحر، وإنهيبين ب، باستخدام تقنية مقايسة الامتصاص المناعي المرتبط بالإنزيم (**ELISA**)، وقياس هرمون البرولاكتين، والهرمون الملوتن (**LH**)، والهرمون المنبه للجريب (**FSH**) باستخدام تقنية **TOSOH**. حُسبت نسبة **LH/FSH**. عدد المرضى في الدراسة الحالية ٦٢ امرأة.

النتائج: كان متوسط (±SEM) لمستويات هرمون التستوستيرون الحر في مصل مرضى متلازمة تكيس المبايض من النمط الظاهري (أ) أعلى بشكل ملحوظ من متوسط المجموعة الضابطة (p=0.001). ومع ذلك، كان متوسط (±SEM) لمستويات هرمون AMH في متلازمة تكيس المبايض من النمط الظاهري (أ) أعلى من متوسط المجموعة الضابطة، ولكنه لم يصل إلى المستوى المعنوي (p=0.06). كان متوسط (±SEM) لمستويات LH ونسبة LH/FSH في متلازمة تكيس المبايض من النمط الظاهري (أ) أعلى بشكل ملحوظ من متوسط المجموعة الضابطة (p=0.03، p=0.001، على التوالي). بالإضافة إلى ذلك، لوحظت ارتباطات مهمة بين الهرمونات المدروسة في متلازمة تكيس المبايض من النمط الظاهري (أ).

الاستنتاج: يُعد النمط الظاهري "أ" النمط السائد بين أنماط متلازمة تكيس المبايض، ويرتبط بارتفاع مستويات هرمون AMH في المصل، والتستوستيرون الحر، والسمنة. قد يُلقي نمط التغيرات الهرمونية وارتباطاتها الضوء على الفسيولوجيا المرضية الجديدة لمتلازمة تكيس المبايض من النمط الظاهري "أ"، وقد يُساعد في استراتيجية العلاج.

الكلمات المفتاحية: هرمون مضاد مولر، التستوستيرون الحر، إنهيبين "ب"، نسبة الهرمون الملوتن/الهرمون المُحفِّز للجريب، متلازمة تكيس المبايض

المؤلف المراسل: زينب جهاد فالح

الايميل: Zz7578942@gmail.com

تاريخ الاستلام: ١٣ **ايلول** ٢٠٢٥

تاريخ القبول: ١١ **تشرين الثاني** ٢٠٢٥

تاريخ النشر: ٢٥ **كانون الأول** ٢٠٢٥

[١] فرع الكيمياء الحيوية السريرية، كلية الطب، جامعة بغداد، بغداد، العراق.

[٢] فرع أمراض النساء والتوليد، كلية الطب، جامعة بغداد، بغداد، العراق.